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Jasmonic acid enhancement of anthocyanin accumulation is dependent on phytochrome A signaling pathway under far-red light in *Arabidopsis*

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ABSTRACT

Anthocyanins are critical for plants. It is shown that the expression of genes encoding the key enzymes such as dihydroflavonol 4-reductase (DFR), UDP-Glc: flavonoid 3-O-glucosyltransferase (UF3GT), and leucoanthocyanidin dioxygenase (LDOX) in anthocyanin biosynthesis pathway is regulated by MYB75, a R2R3 MYB transcription factor. The production of anthocyanin is known to be promoted by jasmonic acid (JA) in light but not in darkness. The photoreceptors cryptochrome 1 (CRY1), phytochrome B (phyB), and phytochrome A (phyA) are also shown to mediate light promotion of anthocyanin accumulation, respectively, whereas their downstream factor COP1, a master negative regulator of photomorphogensis, represses anthocyanin accumulation. However, whether JA coordinates with photoreceptors in the regulation of anthocyanin accumulation is unknown. Here, we show that under far-red light, JA promotes anthocyanin accumulation in a phyA signaling pathway-dependent manner. The *phyA* mutant is hyposensitive to jasmonic acid analog methyl jasmonic acid (MeJA) under far-red light. The dominant mutant of *MYB75*, *pap1-D*, accumulates significantly higher levels of anthocyanin than wild type under far-red light, whereas knockdown of *MYBs* (*MYB75*, *MYB90*, *MYB113*, and *MYB114*) through RNAi significantly reduces MeJA promotion of anthocyanin accumulation. The *phyA pap1-D* double mutant shows reduced responsiveness to MeJA, similar to *phyA* mutant under far-red light. In darkness, a mutant allele of *cop1*, *cop1–4*, shows enhanced responsiveness to MeJA, but *pap1-D* mutant is barely responsive to MeJA. Upon MeJA application, the *cop1–4 pap1-D* double mutant accumulates considerably higher levels of anthocyanin than *cop1–4* in darkness. Protein studies indicate that MYB75 protein is stabilized by white light and far-red light. Further gene expression studies suggest that MeJA promotes the expression of *DFR*, *UF3GT*, and *LDOX* genes in a phyA- and MYB75-dependent manner under far-red light. Our findings suggest that JA promotion of anthocyanin accumulation under far-red light is dependent on phyA signaling pathway, consisting of phyA, COP1, and MYB75.

1. Introduction

Anthocyanins, which are flavonoid compounds, are of pivotal importance to plant growth and development. They attract insects to pollinate flowers [1], disperse seeds and protect plants from damages of ultra violet irradiation [2], insect attack, and pathogen infection [3,4]. The anthocyanin biosynthesis pathway is one of the most studied secondary metabolisms. With the catalyzation by flavonone-3-hydroxylase (F3H), F3′H, and F3′5′H, the biosynthesis of anthocyanins branches into three pathways to form pelargonidin, cyaniding, and delphinidin, respectively, which finally keep constant via UDP-Glc: flavonoid 3-O-glucosyltransferase (UF3GT) [5]. In *Arabidopsis*, the biosynthesis of anthocyanin is regulated by the WD-repeat/bHLH/MYB complex [6], containing TRANSPARENT TESTA GLABRA 1 (TTG1) [7], basic-helixloop-helix (bHLH) transcription factors [8], and R2R3 MYELOBLASTOSIS PROTEIN (MYB) transcription factors [9]. Among the R2R3 MYB transcription factors, MYB75 (also known as PRODUCTION OF ANTHOCYANIN PIGMENT 1, PAP1) is known to promote the expression of genes encoding key anthocyanin biosynthetic enzymes such as dihydroflavonol 4-reductase (DFR), UF3GT, and leucoanthocyanidin dioxygenase (LDOX) [10]. The dominant mutant of *MYB75/PAP1*, *pap1-D*, a T-DNA insertion line that over-expresses *MYB75*, accumulates high levels of anthocyanin [11].

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The production of anthocyanin is promoted by jasmonic acid (JA), an essential phytohormone which is also involved in the protection of plants from pathogenic microorganisms, regulation of plant development and responses to stress stimulation [12–14]. JA signal is perceived by its receptor CORONATINE INSENSITIVE 1 (COI1), an F-box protein [15], which recruits JASMONATE-ZIM-DOMAIN PROTEIN (JAZ) and associates with ARABIDOPSIS SERINE/THREONINE KINASE 1 (ASK1)/ASK2, CULLIN 1 (CUL1), and RING-BOX 1 (RBX1) to assemble SCF^{COI1} complexes for repression of various JA responses via 26S proteasome [15–17]. JAZs also directly interact with MYC2, a MYC-related transcriptional activator with a bHLH motif, to affect JA-regulated biological functions, including inhibition of root growth and photomorphogenesis [18]. It is shown that JA promotes anthocyanin accumulation through the interaction of JAZ proteins and WD-Repeat/bHLH/MYB complexes including MYB75 in light [19].

Anthocyanin biosynthesis is also enhanced by light. As the most important environmental cue, light plays a vital role in regulating plant growth and development [20,21]. Light signal is perceived by multiple photoreceptors, including blue light photoreceptors cryptochromes (CRY) and red/far-red light photoreceptors phytochromes (phyA-E) [22,23]. In *Arabidopsis*, blue light photoreceptor CRY1, red light photoreceptor phyB, and far-red light photoreceptor phyA are known to promote anthocyanin accumulation in blue, red, and far-red light, respectively [22,23]. Phytochromes are well characterized photoreceptors and shown to regulate plenty of developmental processes throughout plant life cycle, such as seed germination, hypocotyls elongation, cotyledon expansion, and flowering [23]. The signaling mechanism of phytochromes involves direct interaction with PHYTOCHROME-INTERACTING FACTORS (PIFs), and promotion of PIFs phosphorylation and degradation via 26S proteasome [24,25]. phyA and phyB are also shown to interact with and inhibit the activity of CONSTITUTIVELY PHOTOMORPHOGENIC 1 (COP1), a RING finger E3 ubiquitin ligase acting as a predominant negative regulator of photomorphogenesis [26,27]. On the other side, COP1 promotes the ubiquitination of phyA to attenuate far-red light signaling [28]. In darkness, *cop1* mutant shows a constitutively photomorphogenic phenotype characterized by shortened hypocotyls, expanded cotyledons, and visible anthocyanin accumulation, which is observed for wild type in light but not in darkness [26].

It has been shown that COP1 physically interacts with MYB75 to promote its degradation via 26S proteasome for the repression of anthocyanin biosynthesis [29], implying that JA and light signaling may utilize this link to regulate anthocyanin accumulation. However, whether JA signaling coordinates with photoreceptor-mediated signaling in the regulation of anthocyanin accumulation is unknown. In this study, we examined the responses of the mutants of critical components in light signaling pathway to jasmonic acid analog MeJA and analyzed the relationship between light and JA signaling at the protein and genetic levels. Our results suggest that under far-red light, JA enhancement of anthocyanin biosynthesis is dependent on phyA signaling pathway, which involves promotion of the expression of genes acting in the late steps of anthocyanin biosynthesis pathway, such as *DFR*, *LDOX*, and *UF3GT*.

2. Materials and methods

2.1. Plant material and growth condition

The growth condition of *Arabidopsis*, including both wild type (Col-0) and mutants were described previously [30]. *Arabidopsis* seeds were sterilized with 20% bleaching water and plated on Murashige and Skoog basal medium (MS, Sigma–Aldrich) with 2% sucrose. After incubated at 4 °C for 3 days, these plants were exposed to white light (150 μmol/m^2/sec) for 12 h, and then transferred to the experimental light conditions.

2.2. Construction of plasmids, mutants, and transgenic lines

Primers (MYBs-miR-1, MYBs-miR-2, MYBs-miR-3, MYBs-miR-4) designed with WMD3 Web microRNA Designer (http://wmd3.weigelworld.org/cgi-bin/webapp.cgi) were used to amplify the amiRNA-*MYBs* (*MYB75*, *MYB90*, *MYB113*, *MYB114*) fragment by overlapping PCR. The resulting fragment was cloned into plant expression vector pHB [31]. The cDNA fragment encoding MYB75 was obtained with primers MYB75F and MYB75R, and then fused to a fragment encoding FLAG tag through PCR and cloned into pHB vector. All the primers are listed in the Table S1. The constructs were transferred into *Agrobacterium tumefaciens* strain GV3101, which was used to transform wild type *Arabidopsis* through floral dip [32]. The *phyA-211*, *cop1–4*, and *pap1-D* mutants were described previously [29,33]. The *phyA pap1-D* and *cop1–4 pap1-D* double mutants were generated by genetic crossing.

2.3. Anthocyanin measurement

Anthocyanin measurement was performed as previously described with minor modifications [34]. 10–20 seedlings were soaked in 600 μl 1% HCl (v/v) diluted with methanol acidified and incubated for 12 h at 4 °C. After addition of 400 μl dd H_2O and 1 ml chloroform, the anthocyanin contents in aqueous phase were measured at 530 nm and 657 nm and calculated using equation: (A530 − A657) / Fw. (Fw represented the total weight of this 10–20 seedlings).

2.4. Protein studies

Western blot assay was described previously [30]. The seedlings were collected in the liquid nitrogen for protein extraction. The samples were fractionated with 10% SDS–PAGE gel and blotted using a PVDF membrane (Amersham Pharmacia). The specific protein was detected by anti-FLAG (home-made, 1:1000 dilution) and anti-Actin (Abmart Inc. China, 1:1000 dilution) antibodies.

2.5. RNA isolation and quantitative RT-PCR

Total RNA was extracted with RNAprep pure plant kit (Tiangen, China), and then reverse-transcribed to cDNA with iScriptTM cDNA Synthesis Kit (BIO-RAD). The qRT-PCR was performed on a CFX96 Real-Time System (BIO-RAD) [33]. *ACTIN8* was used as an internal control, and the primers of the target genes (*DFR*, *LDOX*, *UF3GT*) for qRT-PCR were previously described [19]. The primers used for the examination of *MYBs* (*MYB75*, *MYB90*, *MYB113*, and *MYB114*) genes expression listed in the Table S1.

3. Results

3.1. MeJA promotes anthocyanin accumulation in a phyA-dependent manner under far-red light

Based on the demonstration that JA promotes seedlings anthocyanin accumulation in light but not in darkness [19], we asked whether this process is dependent on photoreceptors. To this end, we analyzed the responsiveness of phyA mutant to various concentrations of the jasmonic acid analog MeJA under far-red light. These results indicate that the wild type (WT) seedlings accumulated significantly more anthocyanin when treated with MeJA at concentrations over 5 μM under far-red light (Fig. 1A and B).

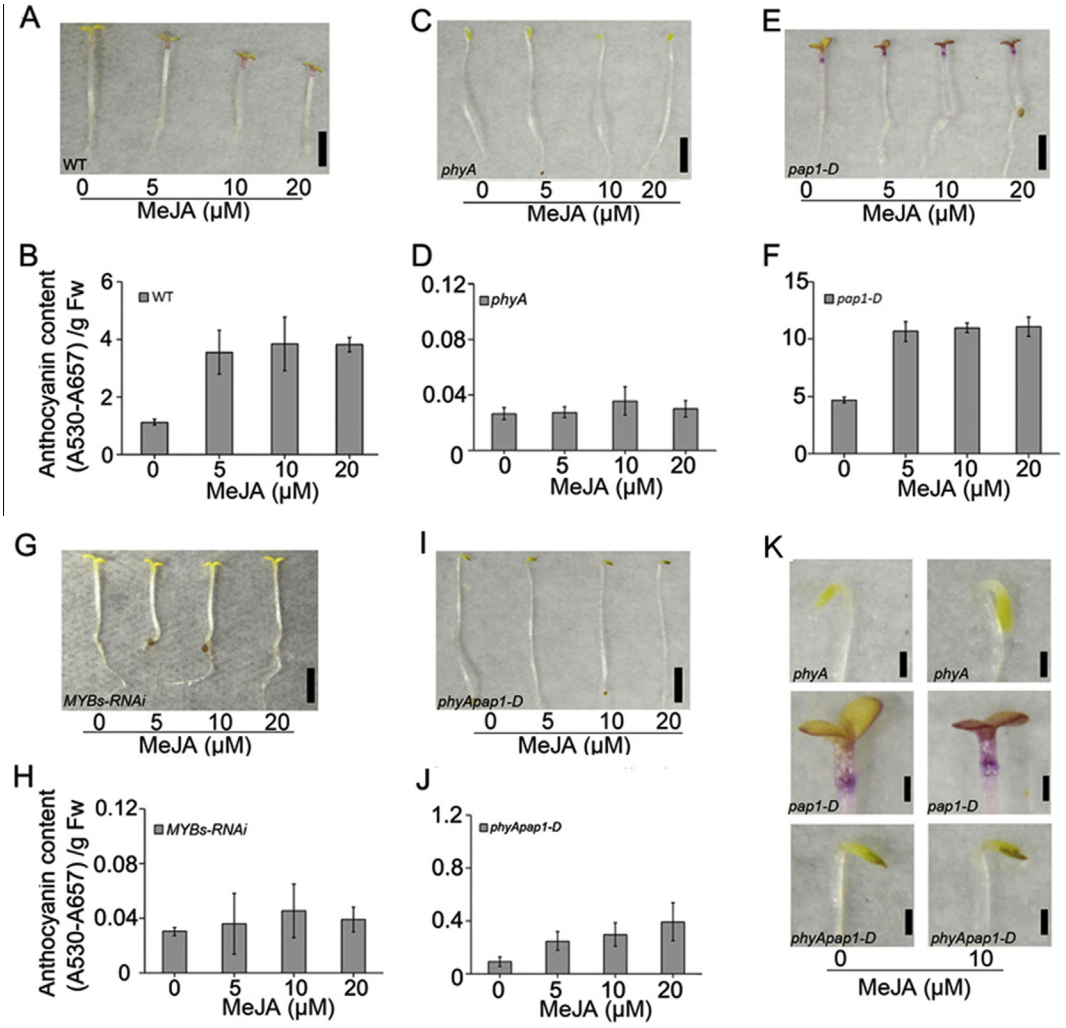

Fig. 1. MeJA promotion of anthocyanin accumulation is dependent on phyA under far-red light. (A, C, E, G, and I) Five-day-old *Arabidopsis* seedlings of WT (A), *phyA* (C), *pap1-D* (E), *35S::amiRNA-MYBs* (*MYBs-RNAi*) (G), and *phyA pap1-D* (I). Seedlings were grown on MS plates supplemented with 0, 5, 10, and 20 μM concentrations of MeJA under 1 μmol m^{-2} s^{-1} far-red light. Scale bars represent 2.5 mm. (B, D, F, H, and J) Bar diagrams represent quantitative anthocyanin contents at different concentrations (0, 5, 10, and 20 μM) of MeJA. The grown condition of seedlings were respectively same as described in (A, C, E, G, and I), Data are presented as mean ± SD, $n = 3$. (K) Enlargement of seedlings cotyledons in (C, E, and I) were grown with 0 and 10 μM MeJA. Scale bars represent 0.6 mm.

However, *phyA* mutant barely accumulated anthocyanin at all the concentrations of MeJA tested, and was basically insensitive to MeJA under far-red light (Fig. 1C and D). Interestingly, in far-red light, the *pap1-D* mutant accumulated significantly higher levels of anthocyanin than WT in the presence or absence of MeJA (Fig. 1E and F). Then, we constructed transgenic *35S::amiRNA-MYBs* plants (Fig. S1A), in which the expression of *MYB75*, *MYB90*, *MYB113*, and *MYB114* was reduced (Fig. S1B). Under white light, the anthocyanin contents in *35S::amiRNA-MYBs* plants were remarkably lower than those in wild type (Fig. S1C). Similar to *phyA* mutant, anthocyanin hardly accumulated in the *35S::amiRNA-MYBs* plants with or without MeJA treatment in far-red light (Fig. 1G and H). We also generated *phyA pap1-D* double mutant by genetic crossing to analyze the genetic interaction of *phyA* and *MYB75/PAP1*. It shows that the *phyA pap1-D* double mutant barely accumulated anthocyanin in the absence of MeJA as well as *phyA* mutant, and accumulated significantly less anthocyanin than *pap1-D* mutant with MeJA treatment at all the concentrations tested (Fig. 1I–K). These results indicate that JA and MYB75 promotion of anthocyanin accumulation is largely dependent on phyA under far-red light.

3.2. MeJA promotion of anthocyanin accumulation is inhibited by COP1 in darkness

It is known that COP1 acts downstream of cryptochromes and phytochromes to repress photomorphogenesis [36,37]. A recent study has demonstrated that COP1 interacts with MYB75 to promote its degradation in darkness via the 26S proteasome pathway [29]. To determine whether COP1 inhibits JA promotion of anthocyanin accumulation, we examined the responsiveness of *cop1–4* to various concentrations of MeJA in darkness. WT seedlings were hardly responsive to MeJA (Fig. 2A and B), whereas application of MeJA at concentrations over 5 μM significantly enhanced anthocyanin accumulation in *cop1–4* mutant (Fig. 2C and D), indicating that COP1 acts to repress JA promotion of anthocyanin accumulation in darkness. Similar to WT, *pap1-D* mutant accumulated low levels of anthocyanin with or without MeJA treatment (Fig. 2E and F). Moreover, anthocyanin accumulation in *35S::amiRNA-MYBs* plants were hardly detectable (Fig. 2G and H). To analyze the genetic relationship between *COP1* and *MYB75/PAP1*, we generated *cop1–4 pap1-D* double mutant, and found that the double mutant accumulated significantly higher levels of anthocyanin than

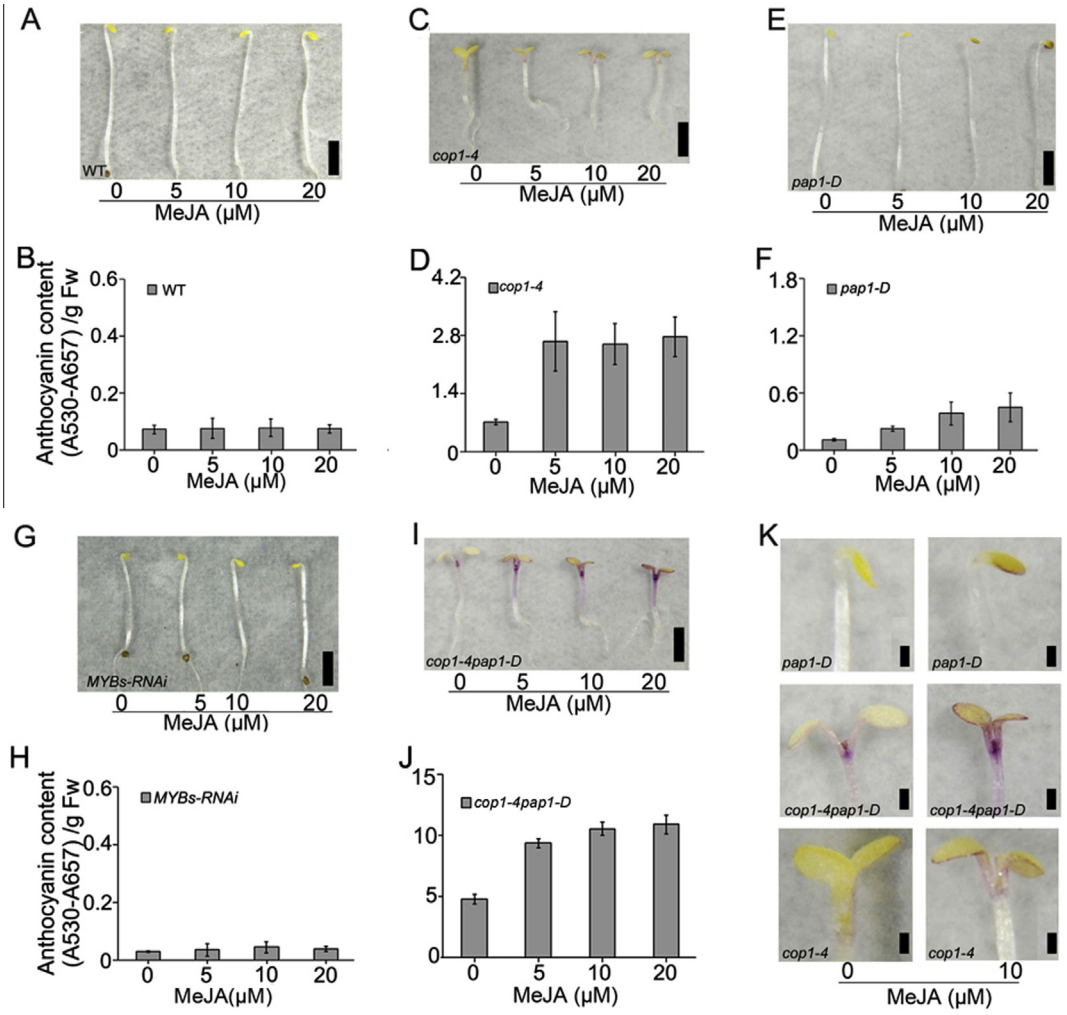

Fig. 2. COP1 represses the promotion of anthocyanin accumulation by MeJA in darkness. (A, C, E, G, and I) Five-day-old *Arabidopsis* seedlings of WT (A), *cop1–4* (C), *pap1-D* (E), *35S::amiRNA-MYBs* (*MYBs-RNAi*) (G), and *cop1–4 pap1-D* (I) Seedlings were grown on MS plates with 0, 5, 10, and 20 μM MeJA in darkness. Scale bars represent 2.5 mm. (B, D, F, H, and J) Bar diagrams represent quantitative anthocyanin contents at different concentrations (0, 5, 10, and 20 μM) of MeJA. The grown condition of seedlings were respectively same as described in (A, C, E, G, and I), Data are presented as mean ± SD, $n = 3$. (K) Enlargement of seedlings in (C, E, and I) grown on plates with 0 and 10 μM MeJA. Scale bars represent 0.6 mm.

cop1–4 or *pap1-D* single mutant (Fig. 2I–K). These results suggest that COP1 inhibition of JA enhancement of anthocyanin accumulation is likely mediated through suppression of MYB75 accumulation in darkness.

3.3. White light and far-red light promote MYB75 accumulation

We explored whether far-red light regulation of anthocyanin accumulation involves regulation of MYB75 protein levels. To do this, we created transgenic plants over-expressing MYB75 tagged by FLAG (*35S::FLAG-MYB75*) and analyzed MYB75 accumulation in three independent transgenic *35S::FLAG-MYB75* lines under white light. As shown in Fig. 3A, 3–10 h white light irradiation clearly promoted MYB75 accumulation in these lines. We then analyzed the accumulation of MYB75 in transgenic *35S::FLAG-MYB75* seedlings upon far-red light illumination, and found that 3 and 10 h far-red light irradiation promoted MYB75 accumulation (Fig. 3B), albeit less pronounced compared to white light treatment. These results indicate that white light and far-red light are able to stabilize MYB75 protein.

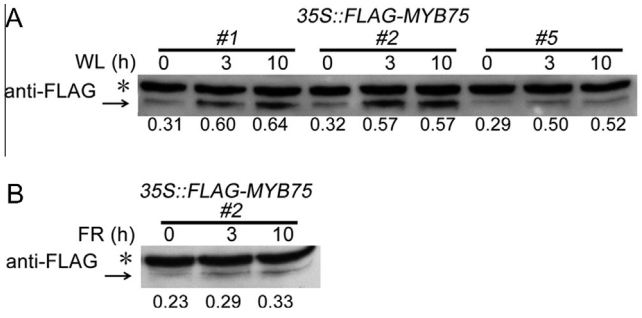

Fig. 3. The protein levels of FLAG-MYB75 are regulated by far-red light. (A) FLAG-MYB75 protein accumulation in three homozygous transgenic *Arabidopsis 35S::FLAG-MYB75 #1, #2* and *#5* lines detected by anti-FLAG using Western blot. Seedlings were grown in darkness for 4 days and then were exposed to 150 μmol m^{-2} s^{-1} white light for 3 and 10 h. "*" represents non-specific bar used as internal reference. The ratio indicates the relative band intensities of FLAG-MYB75 normalized to non-specific bar. (B) FLAG-MYB75 protein accumulation in *35S::FLAG-MYB75 #2* determined with anti-FLAG by Western blot. Seedlings were grown in darkness for 4 days and then were exposed to 1 μmol m^{-2} s^{-1} far-red light for 3 and 10 h. "*" represents non-specific bar used as internal reference. The ratio indicates the relative band intensities of FLAG-MYB75 normalized to non-specific bar.

3.4. MeJA enhances anthocyanin biosynthesis gene expression in a phyA-dependent manner under far-red light

It has been reported that the key anthocyanin biosynthesis genes, such as *DFR*, *UF3GT*, and *LDOX*, are upgraded by MYB75 [38]. Meanwhile, JAZ proteins are known to target MYB75 for repression of the expression of these genes [19]. Based on these studies, we predicted that the mechanism underlying the phyA-dependent promotion of anthocyanin accumulation under far-red light by JA might involve promotion of *DFR*, *UF3GT*, and *LDOX* expression. To test this prediction, we investigated *DFR*, *UF3GT*, and *LDOX* expression in wild type, *phyA* mutant, and transgenic *35S::amiRNA-MYBs* seedlings. As shown in Fig. 4A–C, MeJA treatment significantly promoted the expression of *DFR*, *UF3GT*, and *LDOX* in WT, whereas hardly enhanced the expression of these genes in *phyA* mutant. Moreover, the expression of these genes in transgenic *35S::amiRNA-MYBs* seedlings were not affected by MeJA, similar to *phyA* mutant. These results therefore indicate that JA promotion of anthocyanin accumulation involves JA induction of *DFR*, *UF3GT*, and *LDOX* expression dependent on phyA and MYB75 under far-red light.

4. Discussion

Light is essential for plants to synthesize anthocyanin, and JA promotes anthocyanin accumulation in light but not in darkness [19,35]. To explore how JA promotion of anthocyanin production is dependent on light signaling, we examined the role for phyA in mediating JA promotion of anthocyanin accumulation under far-red light. Our results demonstrate that JA promotion of anthocyanin production is dependent on phyA signaling pathway consisting of phyA, COP1, and MYB75/PAP1. Moreover, we also indicate that both white light and far-red light promote MYB75 accumulation. Besides, our findings showed that JA promotion of *DFR*, *LDOX*, and *UF3GT* expression is dependent on phyA and MYB75 under far-red light. Based on our results, we conclude that photoexcited phyA facilitates the promotion of anthocyanin accumulation by JA by enhancing MYB75 abundance and increasing *DFR*, *LDOX*, and *UF3GT* expression under far-red light (Fig. 4D).

In our study, we confirmed that JA fails to promote anthocyanin accumulation in the dark. Since COP1 is shown to negatively regulate anthocyanin accumulation in darkness [29], we explored the effects of mutations in COP1 and MYB75 on JA enhancement of anthocyanin accumulation and found that the *cop1–4 pap1-D* double mutant accumulated significantly more anthocyanin than either *cop1–4* or *pap1-D* single mutant. Given that COP1 promotes MYB75 degradation via 26s proteasome and that JA promote anthocyanin accumulation through JAZs transcriptional repression of MYB75 [19], we propose a working model (Fig. 4D), in which phyA-COP1 interaction may suppress COP1 activity, resulting in MYB75 accumulation under far-red light. Moreover, JA signaling, perceived by its receptor COI1, inhibits JAZ accumulation, leading to positive effects on MYB75 transcriptional activity.

In the present study, we focused on the exploration of the cross-talk of JA and far-red light in the regulation of anthocyanin accumulation. Our results explain why JA promotes anthocyanin accumulation in light but not in darkness. Namely, JA promotion of anthocyanin production is dependent on the availability of MYB75 protein, whose accumulation is positively regulated by far-red light signaling pathway consisting of phyA, COP1, and MYB75. Based on our findings and previous studies showing that CRY1- and phyB-mediated blue and red light signaling promote anthocyanin accumulation and that the signaling mechanism of both CRY1 and phyB involve direct interaction with COP1 [37,39], we speculate that JA may promote anthocyanin accumulation in blue and red light dependent on the CRY1-COP1-MYB75 and phyB-COP1-MYB75 signaling modules, respectively. It will be worth exploring these speculations in future studies.

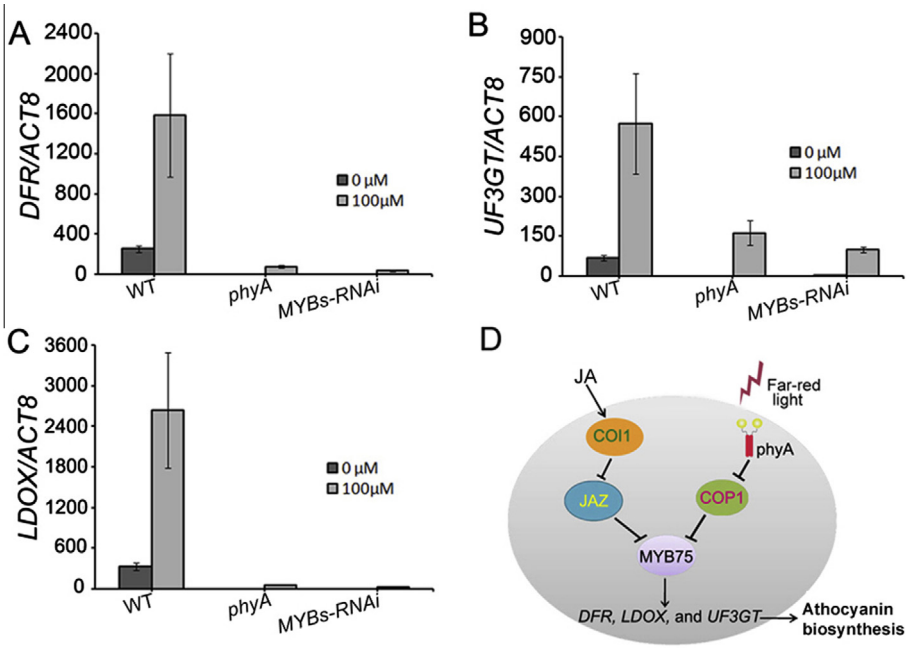

Fig. 4. MeJA induction of anthocyanin biosynthesis genes expression is dependent on phyA under far-red light. (A–C) Relative anthocyanin biosynthesis genes *DFR* (A), *UF3GT* (B), and *LDOX* (C) expression under far-red light grown WT, *phyA*, and *35S::amiRNA-MYBs* (*MYBs-RNAi*) plants. Seedlings were grown for 2 days under continuous white light and 2 days in darkness, and then were transferred to liquid MS media with ethyl alcohol or 100 μM MeJA and exposed to far-red light for 2 h. Data are presented as mean ± SD, $n = 3$. (D) A model showing JA signaling association with phyA signaling to regulate anthocyanin biosynthesis under far-red light. JA signal perceived by COI1 represses JAZs stability and enhances MYB75 transcriptional activity, resulting in up-regulation of *DFR*, *LDOX*, and *UF3GT* expression. Far-red light signal perceived by phyA inhibits COP1 activity, leading to MYB75 accumulation. T bar and arrow indicate the negative and positive regulation, respectively.

Acknowledgments

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.bbrc.2014.10.059.

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